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10/527,950	09/30/2005	Timothy P. Tully	17VV-137227	1059

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EXAMINER	
DUTT, ADITI	

ART UNIT	PAPER NUMBER
1649	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/527,950	TULLY ET AL.	
	Examiner	Art Unit	
	Aditi Dutt	1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *02 March 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-4,6-10,12-15,17-22,24 and 25* is/are pending in the application.

4a) Of the above claim(s) *17-18,25* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4,6-10,12-15,19-22 and 24* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 2 March 2009 has been entered.

Status of Claims

2. The amendment filed on 29 December 2008 has been entered into the record and has been fully considered.

3. Claims 1-4, 6-10, 12-15, 19-22 and 24, drawn to a method of identifying candidate compounds for enhancing CREB pathway function and assessing the effect on CREB-dependent gene expression, are under consideration in the instant application.

Claim Objection

4. Claims 1 and 24 are objected to because of the following informalities:

(i) Claim 1: has a typo in step 'c)' line 1, that recites "….determined in step c)" instead of "b)".

(ii) Claim 24: depends from a cancelled claim 23.

Appropriate correction is required.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

5. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of

35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

6. Claims 1, 3-4, 6-7, 9-10, 12, 14-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over Sheriff et al., (Reg Pept 75-76: 309-318, 1998), in view of Herzog et al. (PNAS 89: 5794-5798, 1992).

7. The claims are directed to a method of identifying candidate compounds for enhancing CREB pathway function, (i) by contacting host cells comprising an indicator gene linked to a CRE promoter with a test compound and CREB function stimulating agent (forskolin); (ii) determining the indicator activity and comparing the same in the above cells versus control cells contacted with the CREB function stimulating agent but not the test agent; (iii) selecting the test compound if the indicator activity in cells treated with CREB function stimulating agent but not the test agent is increased relative to the above control cells; (iv) selecting the test compound if the indicator activity in control cells not treated with the CREB function stimulating agent but with the test agent is not significantly different from the activity elicited by control cells neither treated with the test agent nor with the CREB function stimulating agent; (v) repeating the above steps with a range of concentrations of the test agent; wherein the host cells are neuroblastoma cell, and the indicator gene is luciferase (claims 1, 3-4, 6). The claims further recite identifying and confirming the candidate compound by assessing the effect on CREB-dependent gene expression by contacting cells of neural origin with the candidate compound and CREB function stimulating

agent, assessing and comparing the difference in the endogenous CREB dependent gene expression in the above cells versus that observed in control cells that have been contacted with the CREB function stimulating agent but not with the candidate compound, wherein the cells are hippocampal neurons (claim 7, 9, 10, 12, 14, 15).

Range of concentration:

8. Sheriff et al. teach a method to determine the neuropeptide Y or NPY induced effect on CREB pathway function, such as CREB phosphorylation and CaM kinase activity. Sheriff et al. specifically teach the transfection of cells from the neuroblastoma cell line SK-N-BE2 with a fusion gene containing the CRE site linked to firefly luciferase (indicator) gene. The reference further teaches that the treatment of the transfected cells with NPY and forskolin results in a significant increase of luciferase activity as compared to control cells treated with forskolin but not with the NPY (Abstract; Results: Section 3.5; Figure 5, 5^{th} and 6^{th} bar). Furthermore, Sheriff et al. demonstrate that the luciferase activity in control cells treated with NPY but not with forskolin is not significantly different from the activity elicited by control cells that are not treated with either forskolin or with NPY (Figure 5, 1^{st} and 2^{nd} bar). Sheriff et al further teach that NPY exerts several biological and cognitive functions (e.g. modulation of memory, inhibition of anxiogenic activity, feeding behavior, etc.), by activating the CREB pathway.

9. Sheriff et al. do not teach repeating the method steps with a range of concentrations of the compound or NPY.

10. However, since the disclosure does not specify criticality of the claimed range of doses, optimization within prior art conditions or through routine experimentation is obvious to one skilled in the art.

As stated in MPEP 2144.05:

"The differences in concentration will not support the patentability of subject matter encompassed by the prior art unless there is evidence indicating such concentration is critical. "[W]here the general conditions of a claim are disclosed in the prior art, it is not inventive to discover the optimum or workable ranges by routine experimentation." "The normal desire of scientists or artisans to improve upon what is already generally known provides the motivation to determine where in a disclosed set of percentage ranges is the optimum combination of percentages". *In re Aller, 220 F.2d 454, 456, 105 USPQ 233, 235 (CCPA 1955); Peterson, 315 F.3d at 1330, 65 USPQ2d at 1382; Merck & Co. Inc. v. Biocraft Laboratories Inc., 874 F.2d 804, 10 UDPQ2d 1843 (Fed. Cir.).*

11. It would have been, therefore, obvious to the person of ordinary skill in the art at the time the claimed invention was made to determine the optimal range of doses of the test compound to be added to neuroblastoma cells for identifying compounds such as NPY that enhance the CREB pathway function, as taught by Sheriff et al. The person of ordinary skill in the art would have been motivated to perform such tests to assess the optimum dose of NPY required for augmenting forskolin induced effect on CREB pathway function, because NPY is known to exert several biological functions in the central and peripheral nervous system through induction of CRE binding activity in the hypothalamus. Additionally because of the involvement of CREB pathway in multiple biological and cognitive functions as stated above, the skilled person would be motivated to screen for multiple compounds. The person of ordinary skill in the art would have expected

success because the method assessing in vitro activity of regulatory peptides as a part of screening for candidate compounds for drug development for example, was being experimented and performed in the scientific and medical community, at the time the invention was made.

Gene Expression:

12. Sheriff et al. also teach contacting cells (not transfected) of the neuroblastoma cell line with NPY and forskolin, and assess the expression of endogenous gene containing CRE (Y1 receptor). The reference further teaches that although NPY treatment increases the Y1 mRNA expression, forskolin is more potent in upregulating Y1 receptor message, when compared to untreated controls (page 314, Section 3.6 of Results; Figure 6).

13. Sheriff et al. do not teach the addition of both NPY and forskolin to neuroblastoma cells for the analysis of gene expression.

14. However, in the absence of unexpected results, it would have been prima facie obvious to one of ordinary skill in the art to combine the teachings of the reference and to treat the cells with both NPY (as the candidate compound) and forskolin. Each of these compounds had been taught by the prior art to augment CREB dependent gene expression and increase Y1 mRNA. The instant situation is amenable to the type of analysis set forth in *In re Kerkhoven,205 USPQ 1069 (CCPA 1980)* wherein the court held that it is prima facie obvious to combine two compositions each of which is taught by the prior art to be useful for the same purpose in order to for a third composition that is to be used for the very same

purpose since the idea of combining them flows logically from their having been individually taught in the prior art. Applying the same logic to the instant process claims, given the teaching of the prior art of processes wherein the Y1 gene expression is enhanced by NPY and by forskolin individually, it would have been obvious to contact cells with both NPY and forskolin because the idea of doing so would have logically followed from their having been individually taught in the prior art to be useful as compounds for the same purpose of enhancing CREB dependent gene expression. One of ordinary skill in the art would have reasonably expected to enhance Y1 gene expression upon treating cells with either or both of these compounds since both had been demonstrated in the prior art to elicit the same result.

Hippocampal neurons:

15. Sheriff et al. do not teach contacting cells that are *hippocampal neurons*. Sheriff et al. also do not teach screening a plurality of compounds.

16. Herzog et al teach that the Y1 receptor gene was cloned from a cDNA library derived from the human adult hippocampus (Materials and Methods, para 2, page 5794). Herzog et al. also teach the functional diversity of NPY in various cell types that would be a good resource "to facilitate the development of therapeutic drugs" to combat the different pathology associated with NPY-Y1 receptor and the CREB pathway (concluding para).

17. However, as in case of neuroblastoma, the hippocampus comprise cells of neural origin comprising neurons and absent evidence to the contrary, contacting

of NPY and forskolin to upregulate Y1 gene expression in *hippocampal neurons* would be obvious to one skilled in the art in view of Sheriff et al., and Herzog et al. Furthermore, in considering the disclosure of a reference, it is proper to take into account not only specific teaching of the reference but also the inferences which one skilled in the art would be reasonably be expected to draw therefrom (*In re Preda*, 401 F.2d 825, 159 USPQ 342, 344 (CCPA 1968)). Also, a reference must be considered, under 35 U.S.C. 103, not only for what it expressly teaches but also for what it fairly suggests; all disclosures of prior art, including unpreferred embodiments, must be considered in determining obviousness (*In re Burckel* 201 USPQ 67 (CCPA 1979)).

18. It would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method for elevating Y1 message in neuroblastoma cells as taught by Sheriff et al., by using *hippocampal neurons*. The person of ordinary skill in the art would have been motivated to make that modification and would have expected success because Y1 is present in the hippocampus as demonstrated by Herzog et al. Based upon the functional diversity of the NPY-Y1 receptor coupled to various second messenger system in different cells as taught by Herzog et al., the person of ordinary skill would be certainly motivated to try screening for various compounds for the development of different therapeutic drugs with reasonable success. Therefore, the claimed invention as a whole was clearly *prima facie* obvious over the prior art.

19. Claims 19(a-k) and 20-22 and 24 are rejected under 35 U.S.C. 103(a) as being unpatentable over Sheriff et al., (Reg Pept 75-76: 309-318, 1998), in view of Herzog et al. (PNAS 89: 5794-5798, 1992), and further in view of Barad et al. (PNAS 95: 15020-15025, 1998).

20. The claims are directed to a method of identifying candidate compounds for enhancing CREB pathway function, (i) by contacting host cells comprising an indicator gene linked to a CRE promoter with a test compound and CREB function stimulating agent; (ii) determining the indicator activity and comparing the same in the above cells versus control cells contacted with the CREB function stimulating agent but not the test agent; (iii) selecting the test compound if the indicator activity in cells treated with CREB function stimulating agent but not the test agent is increased relative to the above control cells; (iv) selecting the test compound if the indicator activity in control cells not treated with the CREB function stimulating agent but with the test agent is not significantly different from the activity elicited by control cells neither treated with the test agent nor with the CREB function stimulating agent; (v) repeating the above steps with a range of concentrations of the test agent; wherein the host cells are neuroblastoma cells, the CREB function stimulating agent is forskolin and the indicator gene is luciferase. The claims further recite identifying and confirming the candidate compound by assessing the effect on CREB-dependent gene expression by contacting hippocampal cells with the candidate compound and CREB function stimulating agent, assessing and comparing the difference in the

endogenous CREB dependent gene expression in the above cells versus that observed in control cells that have been contacted with the CREB function stimulating agent but not with the candidate compound, wherein the compound is selected if the gene expression in the above treated cells is increased relative to control groups matching treatment criteria as in (ii), (iii) and (iv).

21. The teachings of Sheriff et al. and Herzog et al are set forth above.

22. Sheriff et al. and Herzog et al. do not teach changes in gene expression after the addition of both forskolin and test agent/candidate compound to the hippocampal neurons.

23. Barad et al. teach a method of elevating cAMP concentrations, using hippocampal slices from mice, that were incubated with increasing range of 4 concentrations of the phosphodiesterase type IV inhibitor, rolipram (0, 0.03, 0.3 and 3 μM) in the presence or absence of the adenylyl cyclase activator forskolin (abstract, page 15021). The reference also demonstrates that cAMP levels at lower concentrations of rolipram (up to 0.3 uM) were not significantly different from the basal level (without rolipram and forskolin) (page 15021, 'results' para 1, Figure 1). Furthermore, the results demonstrate that the endogenous cAMP levels in cells contacted with rolipram and forskolin are increased relative to the levels in the control group treated with forskolin but not rolipram. It is noted that forskolin is an adenylyl cyclase activator as stated above, which would inherently increase endogenous cAMP concentrations via increased adenylyl cyclase expression as evidenced by Insel et al (Cell Mol Neurobiol 23: 305-314, 2003,

abstract). It is also well known that adenylyl cyclase is a positive effector of CREB, inducing CREB dependent gene expression.

24. As already explained above, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method for elevating Y1 message in neuroblastoma cells as taught by Sheriff et al., by using *hippocampal neurons*. Likewise, it would have also been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method for elevating Y1 message in neuroblastoma cells as taught by Sheriff et al., by elevating cAMP levels from hippocampal slices as taught by Barad et al. The person of ordinary skill in the art would have been motivated to substitute hippocampal neurons for hippocampal slices, because neurons are individual cells that would elicit the CREB dependent gene expression in a cell specific manner. The person of ordinary skill would make that modification with a reasonable expectation of success, because gene expression studies using individual neurons were well established at the time of filing of the instant invention.

25. Thus, the claimed invention as a whole was *prima facie* obvious over the combined teachings of the prior art.

26. Claims 2, 8 and 13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Sheriff et al., (Reg Pept 75-76: 309-318, 1998), in view of Herzog et al. (PNAS 89: 5794-5798, 1992), and further in view of Barad et al. (PNAS 95: 15020-15025, 1998).

27. The claims recite that the host cells or the cells of neural origin are contacted with the test/candidate compound prior to contact with the CREB function stimulating agent.

28. The teachings of Sheriff et al., Herzog et al. and Barad et al. are set forth above.

29. Sheriff et al., Herzog et al. and Barad et al. do not teach that the cells are contacted with the compound before contacting the CREB stimulating agent.

30. However, since the disclosure does not specify criticality of the claimed sequence of addition of the test/candidate compound and the CREB function stimulating agent, optimization within prior art conditions or through routine experimentation is obvious to one skilled in the art.

31. It would have been, therefore, obvious to the person of ordinary skill in the art at the time the claimed invention was made to determine the sequential addition of compounds to be added to neuroblastoma cells or hippocamal cells for identifying compounds that enhance the CREB pathway function, as taught by Sheriff et al. or Barad et al. The person of ordinary skill in the art would have been motivated to perform such tests, to assess the optimum conditions to achieve the desirable increase in CREB function. The person of ordinary skill in

the art would have expected success because in vitro cell based assays using one or more agents were routinely performed in the scientific and medical community, at the time the invention was made.

32. Thus, the claimed invention as a whole was *prima facie* obvious over the combined teachings of the prior art.

Applicant's Remarks

Sheriff in view of Herzog

33. Applicant traverses the rejection alleging that Sheriff et al do not "characterize the results" the way "the Examiner's own improper independent analysis of the data" is presented. Specifically Applicant asserts that contrary to Examiner's assertion, Sheriff teaches that forskolin and NPY increase luciferase activity, and that this "may involve more than one member of CRE binding transcription factors". Additionally, Applicants allege that Examiner's conclusion that because CREB and NPY have known cognitive modulatory effects, "screening compounds for such function" is obvious and a motivation to the person of ordinary skill, is absolutely "ungrounded" and "simply impermissible". Also, Applicants continue to argue that Sheriff does "not teach or suggest screening a plurality of compounds for potential development as candidate cognitive enhancer compounds". Applicant further argues that Examiner's proposition on "hindsight reasoning" is "inapposite to the present facts", because

Examiner has not identified any reason for the teaching of "a plurality of test compounds" in the prior art. Furthermore, Applicant alleges that the Examiner's comments on this limitation being in the preamble, and that testing a plurality of test compounds is required by screening, validates that the references do not "support a prima facie case of obviousness".

34. Applicant's arguments are fully considered, however, are not found to be persuasive. As stated in the previous Office Action, it is argued that Sheriff et al. demonstrate that the luciferase activity in control cells treated with NPY but not with forskolin is not significantly different from the activity elicited by control cells that are not treated with either forskolin or with NPY (Figure 5, 1st and 2nd bar). Additionally, the treatment of the transfected cells with NPY and forskolin results in a significant increase of luciferase activity as compared to control cells treated with forskolin but not with the NPY (Abstract; Results: Section 3.5; Figure 5, 5th and 6th bar). Applicant's allegation that Sheriff's teaching is contrary to Examiner's interpretation is mistaken because of comparable observations matching the requirements in the claimed method as shown in Figure 5. Furthermore, Sheriff's analysis of data showing that both forskolin and NPY increased luciferase activity is in reference to Figure 4A (see page 313, col 2, para 2). Figure 4A does not fully represent the claimed invention because it does not have the experimental group demonstrating the indicator activity in the presence of both forskolin and NPY. Moreover, the claims do not require that the test substance is not involving more than one member of CRE binding

transcription factors, thus Applicant's arguments emphasizing this reasoning is irrelevant. Furthermore, it is reiterated that because CREB and NPY are important regulators in cognitive function and because the combined references teach that NPY stimulates the CREB pathway, a person of ordinary skill would be motivated to test for more enhancer compounds like NPY.

35. With regards to Applicant's arguments on "plurality of test compounds", it is emphasized that knowing the diverse functional characteristics of the CREB pathway and molecules associated with the same, coupled with the prior art guidance for the method steps of the claimed screening protocol, it would be not only be obvious for a skilled artisan to perform the experiment once, but be motivated to do repititively with multiple or a "plurality of compounds". Furthermore, it is reiterated that "screening a plurality of compounds" is merely part of the preamble of the claim reciting the screening method that reflects a general feature of any screening protocol without providing further limitations to the invention. The addition of the limitation "plurality of compounds" does not add any distinct limitation to the claimed method, rather is redundant. If the body of a claim fully and intrinsically sets forth all of the limitations of the claimed invention, and the preamble merely states, for example, the purpose or intended use of the invention, rather than any distinct definition of any of the claimed invention's limitations, then the preamble is not considered a limitation and is of no significance to claim construction. *Pitney Bowes, Inc. v. Hewlett-Packard Co.*,

182 F.3d 1298, 1305, 51 USPQ2d 1161, 1165 (Fed. Cir. 1999). See also *Rowe v. Dror*, 112 F.3d 473, 478, 42 USPQ2d 1550, 1553 (Fed. Cir. 1997).

36. Furthermore, in response to Applicant's allegations for "hindsight" reasoning, it is reiterated that "[a]ny judgement on obviousness is in a sense necessarily a reconstruction based on hindsight reasoning, but so long as it takes into account only knowledge which was within the level of ordinary skill in the art at the time the claimed invention was made and does not include knowledge gleaned only from applicant's disclosure, such a reconstruction is proper." *In re McLaughlin* 443 F.2d 1392, 1395, 170 USPQ 209, 212 (CCPA 1971).

37. Thus, contrary to Applicant's contention, a proper prima facie case of obviousness was established and the claimed invention stand rejected over the combined teachings of the prior art.

Sheriff in view of Herzog and in further view of Barad

38. Applicant maintains the prior arguments stating that neither Sheriff nor Herzog teaches or suggests screening a plurality of compounds to determine the ability of compounds to enhance the CREB pathway and identify the compounds as a candidate cognitive enhancer compound based on the claimed invention, nor does Barad remedy the deficiencies.

39. Applicant's arguments are fully considered but not found to be persuasive for reasons explained above. As stated in the previous Office Action it is

reiterated that it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method for elevating Y1 message in neuroblastoma cells as taught by Sheriff et al. by using hippocampal neurons, or hippocampal slices as taught by Barad et al. and be motivated to do so because neurons are individual cells that would elicit the CREB dependent gene expression in a cell specific manner.

Sheriff in view of Herzog and in further view of Barad

40. Applicant maintains the same argument as stated in the previous response that neither Sheriff nor Herzog teaches or suggests screening a plurality of compounds to determine the ability of compounds to enhance the CREB pathway and identify the compounds as a candidate cognitive enhancer compound based on the claimed invention, nor does Barad remedy the deficiencies.

41. Applicant's arguments have not been found to be persuasive for reasons explained above.

Conclusion

42. No claims are allowed.

43. This is a RCE of applicant's earlier Application No. 10/527,950. All claims are drawn to the same invention claimed in the earlier application and could have been finally rejected on the grounds and art of record in the next Office action if

they had been entered in the earlier application. Accordingly, **THIS ACTION IS MADE FINAL** even though it is a first action in this case. See MPEP § 706.07(b). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

44. A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no, however, event will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

45. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Aditi Dutt whose telephone number is 571-272-9037. The examiner can normally be reached on M-F.

46. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffery Stucker, can be reached on 571-272-0911. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

47. Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information

for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov/. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

AD
14 May 2009

/Jeffrey Stucker/

Supervisory Patent Examiner, Art Unit 1649